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Common krait (*Bungarus caeruleus*) bite in Anuradhapura, Sri Lanka: a prospective clinical study, 1996–98

S A M Kularatne

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S A M Kularatne, General Hospital, Anuradhapura, Sri Lanka

Correspondence to: Dr S A M Kularatne, Department of Medicine, Faculty of Medicine, Peradeniya University, Peradeniya, Sri Lanka; samk@pan.lk

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Common krait (*Bungarus caeruleus*) is the deadliest snake found commonly in the dry zone of Sri Lanka. In Anuradhapura, 210 farmers bitten by the common krait over a three year period were investigated prospectively from 1 January 1996. The sex ratio was equal, 110 (52%) patients were in the age group 10–30 years. One hundred and one (48%) patients were severely envenomed and needed mechanical ventilation from 12 hours to 29 days (mode two days). The bite occurred at night while the victims were asleep on the floor. In 99 (47%) situations killed specimens were available for identification. The cardinal symptom was abdominal pain developing within hours of the bite. Alteration in the level of consciousness was observed in 150 (71%) patients: drowsy in 91 (43%), semiconscious in 24 (11%), and deep coma in 35 (17%). Autonomic disturbances included transient hypertension, tachycardia, lacrimation, sweating, and salivation. These manifested in 139 (66%) patients with moderate to severe envenomation. One hundred and forty nine (71%) had hypokalaemia and 105 (50%) metabolic acidosis, anterograde memory loss in 84 (40%), and delayed neuropathy in 38 (22%) patients. Polyvalent antivenom had no significant benefit ($t = 0.5$) in reversing respiratory paralysis and preventing delayed neurological complications. Sixteen (7.6%) patients died and a submucosal haemorrhage in the stomach was seen at necropsy in three cases. Mortality could be minimised with early and free access to mechanical ventilation.

The common krait (*Bungarus caeruleus*, Schneider 1801) is a proteroglyphous elapid snake commonly found in Sri Lanka, Pakistan, Bangladesh, and India.[1–3] The highest incidence of bites in Sri Lanka was reported from the North Central Province, where the vegetation and climate provide an ideal habitat for snakes.[4–6] Common victims of *B caeruleus* are farmers who live in open wattle-and-daub houses and farmers sleeping in watch huts in agricultural fields.[6–8] A significant number of patients die before reaching hospital.[6 9] The approximate incidence of common krait bite in the late 1970s was 8.6% in Sri Lanka; the remainder were bitten by the hump nosed viper (27%), Russell's viper (17.5%), and cobra (12%).[10] Surveys carried out in 1983 in the Anuradhapura district have shown that *B caeruleus* bites accounted for 45% of 110 deaths in the field due to snake bites.[9] However in recent years more patients have sought the life saving benefits of hospital treatment and the case fatality has come down significantly with assisted ventilation.[7 8 11] The counterpart, the Ceylon krait (*Bungarus ceylonicus*, Gunther 1864) is the indigenous species found upcountry and in the wet zone of Sri Lanka, but seldom bites man.[1 12] Historically, both species are reputed to be the most poisonous snakes in the island.[1 13]

The aim of the study was to describe the epidemiology, define clinical features, study acute complications, delayed manifestations and outcome, analyse the efficacy of polyvalent antisera, study the pathophysiology, and define optimum intensive care management of common krait envenoming.

METHODS

The prospective study included all the admissions with common krait bite to the General Hospital, Anuradhapura, Sri Lanka, from 1 January 1996 to the end of 1998. This institution is the main referral centre situated in the central dry zone of the island and has facilities for assisted ventilation and intensive care. The offending snakes were identified by either studying characteristics of the dead snake if it was produced, or showing the patient specimens of formalin preserved snakes. If both failed, clinical features and circumstantial evidences were used for arriving at the diagnosis of common krait bite. Patients were assessed at the time of admission and periodically, until the final outcome.

Clinical assessment included history and examination for neurotoxicity: ptosis, eye movements, papillary size and reaction to light, power of neck flexors and limb muscles, respiratory rate, tidal volume, chest expansion, strength of speech, level of consciousness, brain stem reflexes and cardiac status: pulse rate, blood pressure; muscle tenderness, and local effects. Further assessments included measurement of urine output, whole blood clotting test at 20 minutes, serum electrolytes, blood urea, serum creatinine, blood gases, and a 12 lead electrocardiogram. Limited necropsies were done on death. Neurophysiological tests (nerve conduction velocity of median, ulnar, sural, common peroneal, and posterior tibial nerves) were done as a routine for management of patients with late onset neuropathy. Their was regular follow up of the survivors.

Severity of neuromuscular paralysis (degree of envenoming) was graded as mild: ptosis and external ophthalmoplegia; moderate: weakness of bulbar and neck muscles but not requiring assisted ventilation; and severe: those requiring assisted ventilation due to paralysis of respiratory muscles. Grading of muscle weakness was from grade 0–5 on the scale of the British Medical Research Council.[14]

Alterations in level of consciousness was arbitrarily classified as normal, drowsy, semicoma, and deep coma.[14] The grade of deep coma consisted of patients who went into a state of total unresponsiveness to deep pain and to loud command, having fixed dilated pupils and absent brain stem reflexes. The grade drowsy consisted of patients having rational motor and

verbal response, who remained sleepy with intermittent disorientation but were arousable. The balance belonged to the semicoma group.

Autonomic dysfunction parameters included chemosis, lacrimation, salivation, sweating, rate and fluctuation of heart beat, and fluctuation of systolic and diastolic blood pressure. Monitoring pulse rate according to the respiratory cycle, changing position, and pharyngeal stimulation was possible.[14] After reasonable recovery of patients, memory and higher functions were tested according to the standard recommendation.[14]

The routine management protocol of common krait bite in hospital was adopted without modification. Initial resuscitation was the cornerstone of management and patients in severe respiratory paralysis were offered assisted ventilation. Patients with stable respiration were monitored for early neuromuscular respiratory paralysis.

Initiation of assisted ventilation was considered when tidal volume was below 200 ml, the power of neck flexors was below grade 3, there was central cyanosis, mental confusion, and failing speech. With the earliest sign of systemic envenoming, all the patients were treated with Indian polyvalent antivenom (Haffkine Bio-Pharmaceutical Corporation Ltd, Bombay,India) infusion, which is raised against four snakes including the common krait. The standard dose of 10 vials was given to the all patients and it was repeated in 36 cases. Vital parameters such as heart rate and rhythm, peripheral oxygen saturation, maintenance of fluid balance, physiotherapy, adjustment of assisted ventilation to maintain normal blood gases, early detection, and treatment of complications either by a result of envenoming or mechanical ventilation were closely monitored. Details including age, sex, socioeconomical status, time, place and site of bite, and clinical assessment, investigations, complications, outcome, and treatment were documented in a well designed pro forma. Recovered patients were followed up for late sequelae.

RESULTS

There were 210 patients with *B caeruleus* bites, accounting for 9% of all snake bite admissions to the General Hospital, Anuradhapura. Envenoming was severe in 101 (48%), moderate in 38 (42%), and mild in 49 (23%) patients. Interestingly, in 22 (10%) patients there were no signs of envenoming, in spite of proved bites with definitive fang marks. Overall, in 99 (47%) situations, the offending snakes were killed and brought along with the patients and identified as common krait by the author. In the rest, other diagnostic criteria were adopted.

Socioeconomic status, time of bite, and seasonal distribution

All the patients were from poor farming families living in villages, 202 (96%) of them in cadjan thatched, wattle-and-daub houses where individuals sleep on the floor. These houses were surrounded by uncleared vegetation. Most of the bites occurred at night while the victims were sleeping on the floor except in three cases where the bite took place during the daytime on footpaths and in another in a watch hut situated in a treetop. In two thirds, the bite took place between 2200 to 0400 hours, indicating midnight preponderance.

A significant number of patients 65 (31%) had not been aware of the bite but had woken up with colicky abdominal pain. In 35 (17%) patients the site of the bite was undetectable and they presented with abdominal pain, dyspnoea, dysphagia, and signs of neuromuscular paralysis. All detectable bites were seen on exposed parts of the body: 50 (30%) on the hand, 27 (16%) foot, 20 (12%) upper arm, five neck, and three earlobe (out of 168 patients).

The maximum number of krait bites 103 (49%) occurred during the rainy season, especially in the months of September to December, during the North and East monsoon. It was our experience that most admissions of krait bites follow rainfall, even after an isolated shower during the drier months of the year. However, there were 19 admissions during the severe drought in July to August 1997.

Table 1 Incidence of different symptoms and signs on admission

Symptom/sign	All patients (%)	Severe patients (%)*
Dyspnoea	143 (68)	91 (90)
Abdominal pain	143 (68)	83 (82)
Dysphagia	134 (64)	73 (73)
Chest pain	109 (52)	60 (60)
Faintness	96 (46)	46 (46)
Giddiness	67 (32)	32 (32)
Myalgia	63 (30)	36 (36)
Vomiting	33 (16)	18 (18)
Ptosis	147 (70)	83 (82)
Weakness of limbs	134 (64)	78 (78)
Decreased consciousness	134 (64)	78 (78)
Weakness of neck flexors	126 (60)	68 (68)
Blurred vision	111 (53)	65 (65)
Decreasesed respiration	94 (45)	77 (77)
Local reaction	63 (30)	18 (18)

*Ventilated patients.

Age and sex distribution

The sex ratio was equal and the commonly affected age group was between 10 and 30 years (52%). Envenoming was extremely severe in children below 10 years of age (12 patients). Nine patients were above the age of 60 years.

Clinical features

Abdominal pain was the first symptom to manifest with a range of minutes to a few hours.

Other common clinical features were weakness of limbs, inability to stand up, drooping of eye lids, double vision, difficulty in breathing, and changing sensorium; all progressed rapidly to severe neuromuscular paralysis. Less commonly, myalgia, paraesthesia at the site of bite, decreased hearing and vision, and faintness were observed. Very often the site of the bite and fang marks were indistinct and the local reaction was faint. Bites on fingers and hands invariably produced a significant local reaction with swelling and pain (table 1). The time taken for hospital admission from the time of bite ranged from one to 20 hours; 164 patients were admitted to hospital within seven hours of the bite.

Respiratory paralysis

Of the 210 patients, 101 were ventilated, and the time lag to initiation of ventilation was 30 minutes to 50 hours with a mode of six hours. Seventy five (74%) patients needed ventilation within 10 hours of the bite. Fifty five (54%) patients developed respiratory arrest with no recordable tidal volume during ventilation and 13 (12%) of them did so within one hour of admission. Duration of ventilation ranged from 12 hours to 29 days (mode two days).

Recovery of neuromuscular paralysis was assessed by examination of muscle power and measuring tidal volume at regular intervals. Recovery of neck flexion to power grade 2–3 had significant correlation to the onset of stable recovery of respiration ($r = 0.491$, $p<0.05$); this was used as a parameter to wean patients off ventilation.

Level of consciousness

The level of consciousness was normal in 60 (29%), drowsy in 91 (43%), semiconscious in 24 (11%), and deep coma in 35 (17%) patients. Patients in deep coma had absent brainstem and spinal reflexes; pupils remained fully dilated and light reflexes were absent. The onset of deep coma ranged from two

Table 2 Details of autonomic dysfunction (188 patients)

Dysfunction	Severe group (%) (n=101)	Mild–moderate (%) (n=87)
Chemosis, lacrimation, sweating	101 (100)	38 (43)
Heart rate (beats/min)		
90–120	45 (44)	46 (52)
120–150	38 (37)	4
>150	18 (18)	0
Paralytic ileus	42 (41)	0
Blood pressure (mm Hg)		
130/90–150/100	68 (67)	42 (48)
150/110–190/130	24 (23)	0

Table 3 Recovery of functions in severely envenomed patients (n=101)

Recovered function	No of patients	Mean (days)*
Cough reflex	53	2.6
Gag reflex	54	2.8
Normal consciousness	90	2.8
Ophthalmoplegia	101	3.6
Memory	78	4.0
Neck to power grade 2–3	96	4.0
Normal respiration	101	3.0
Facial muscles	101	5.0
Ptosis	101	5.1
Distal muscles (hand grip/foot)	101	6.0
Proximal muscles (hip/shoulder)	101	7.5
Sitting up, unsupported	96	7.0
Neck to full strength	101	8.7

*Mean number of days taken for full recovery of function.

hours after the bite up to 48 hours and the persisted from six hours to five days (mode 12 hours).

Patients in deep coma developed more complications than others during assisted ventilation. These included collapse of lung segments in 10 patients, hypostatic pneumonia in eight, ileus in 23, ventricular arrhythmia in two, atrial tachycardia in 18, and adult respiratory distress syndrome (ARDS) in six patients (five died). Twenty two patients in deep coma made a complete recovery. The level of consciousness had a direct and significant correlation to the duration of respiratory paralysis ($r = 0.6386$, $p<0.01$) as patients in deep coma needed longer duration of ventilation.

Autonomic dysfunction

Fluctuation of sympathetic and parasympathetic activity was observed in all grades of envenoming during the first 48 hours and included sweating, tearing, chemosis, fluctuation of heart rate, and blood pressure. These were more marked in the severely envenomed patients (table 2).

There was no change in heart rate or blood pressure with changes in position or to pharyngeal stimulation during physiotherapy. Paralytic ileus leading to abdominal distension and absent bowel sounds was manifested in 42 patients. Sixty six patients with severe envenoming had semidilated pupils with positive light reflex and the remaining 35 had completely dilated fixed pupils during the stage of deep coma.

Hypokalaemia

Significant hypokalaemia (serum potassium <3.5 mmol/l and U waves on the electrocardiogram) was observed in 149 (71%) patients during early stages, especially in the first 48 hours. Hypokalaemia was not related to respiratory alkalosis and needed replacement therapy, depending on the severity. Furthermore, metabolic acidosis within first 24 hours was seen in 105 (50%) patients (hypokalaemic acidosis).

Specific treatment

As specific antivenom raised against common krait venom was not available, all the patients in the series were given polyvalent Haffkine antivenom in different doses in the first 48 hours, regardless of the severity of the condition. There was no significant difference between the duration of ventilation (recovery of ventilation) and the dose of antivenom administered ($t = 0.5$, $p = 0.15$). This suggests that higher doses of antivenom have no effect in reversing respiratory failure in common krait bite.

Outcome

Highest mortality rate was seen in the year 1996, during which seven (17%) patients died in spite of intensive care management. The causes of death were ARDS in four, arrhythmia in one, and poor resuscitation before admission in two. Three patients developed ARDS while recovering from neuromuscular paralysis and died despite specific management. Retrospectively, three of these patients had had nasal insufflation of herbal medicinal juice (gnasna); this could have induced chemical pneumonits leading to ARDS. Six (7%) patients died in 1997 and five of them were children under 10 years. Outcome in 1998 had improved: three (3.7%) died (ARDS in one and delay in resuscitation in two patients). There were no deaths in children after the availability of a paediatric intensive care unit in close proximity to the children's ward. Overall mortality rate was 7.6%. The recovery rate of physiological functions in severely envenomed patients (n = 101) was assessed (table 3).

Eight necropsies were done on three clinical categories of cases: four cases of ARDS, one case of hypoxic brain death ventilated for more than two days, and three cases of acute death within 17 hours of the bite. In ARDS patients the lungs were congested and haemorrhagic and histology confirmed ARDS; erosions and bleeding points were seen in gastric mucosa, one case showed mottling haemorrhages in the renal cortex and bleeding in both adrenal glands. In the acute deaths large submucosal haemorrhagic patches (5–9 cm in diameter) in the gastric mucosa were seen in all the three cases; mottling haemorrhages in myocardium, adrenal glands, and kidneys were seen in one case.

Congestion of brain, lungs and severe gastric erosions, were seen in the patients with hypoxic brain death.

Anterograde memory loss

Eighty four (40%) patients who recovered had a variable duration of memory loss. The range was 12 hours to eight days (mode three days). Duration of anterograde memory loss had no significant correlation to the lowest level of consciousness ($r = 0.403$, $p = 0.000$) or the duration of respiratory paralysis ($r = 0.751$, $p = 0.000$). It was an independent and constant feature in severe envenoming.

Delayed neuropathy

Thirty eight patients had delayed neurological deficits. Fourteen of them had nerve conduction defects in the ulnar, median, and common peroneal nerves that lasted for two weeks to six months before complete recovery. Sensory loss at the site of bite was observed in 34 patients; this lasted for two weeks to six months. One patient had bilateral ulnar nerve palsy with wasting of small muscles of the hands, four patients had glove type sensory motor neuropathy, and one patient developed cerebellar ataxia which persisted for two years.

DISCUSSION

The medical importance of the genus *Bungarus*, its member species, details of physical characterisation, and distribution

in South East Asia was described in the early 19th century.[1 2] It was believed that the venom of the common krait (*B caeruleus*) produces depression of vital centres in the brainstem and contains haemolysins and haemorrhagins.[1]

Currently it is understood that bungarotoxins block the transmission at the neuromuscular junction.[7 15] The mortality rate of 77% among 35 proved common krait bites in India[16] and deaths of 27 patients who received only traditional treatment in Sri Lanka[5] have been reported in the year 1954 and 1987 respectively. Field surveys and case studies done in the past have described the ecology, epidemiology, and some clinical aspects of common krait bite in Sri Lanka.[3 4 5 7 17] Some studies concluded that 97% of snake bite deaths in Sri Lanka were due to common krait and Russell's viper bites.[10] There were 27 082 snake bites and 164 deaths reported in Sri Lanka in the year 1996.[18] However, incidence and prevalence figures of bites and deaths due to different snakes are not available in the national statistics. Nevertheless, the latest statistics of the General Hospital, Anradhapura, showed the incidence of species specific snake bite as Russell's viper 48%, non-venomous snakes 33%, common krait 9%, hump nosed viper 7%, and cobra 2%. The present study is the largest study of common krait envenoming in Sri Lanka that has unearthed significant findings worth further discussion and debate.

The common krait is a nocturnally active, terrestrial snake which lives close to human dwellings, but is not vicious by nature.[3] It creeps into houses over the ground or through the roof and exhibits arboreal tendencies.[1 3 4 7 13] The present study testifies to these facts but also found the occasional daytime bite and biting in the tops of trees. It has also been observed by others that the bites occur at night while the victims are asleep.[1 3 4 7 8] Why the snake prefers to creep into human dwellings and how man becomes the victim is yet to be documented. The common krait normally prefers to feed on small snakes.[1 3] However, the krait is attracted by mice, rats, and geckos that are abundant in houses.[1 3] While asleep, humans may be bitten either due to accidental handling or rolling over the snake, or exposed parts of the human body might be misidentified as prey. These may be possible provocative factors for a krait bite even though the common krait remains naturally indolent. The seasonal pattern of the common krait bite has been explained with mating behaviour,[1 3 4] but the present study has shown the influence of changing environment like rain and severe drought as contributing factors.

Abdominal pain is an important and unexplained symptom observed in the present study and mentioned by other authors.[7 8 16 17] When a patient is unaware of the bite, and wakes in the night with colicky abdominal pain, mainly in the epigastrium, they may be misdiagnosed due to lack of awareness on the part of medical practitioners. The finding of submucosal haemorrhages in the stomach in acute death may be responsible for this symptom. However, the finding of gastric erosions in many necropsies could be related to acute stress.

Mottling haemorrhages in the other organs and adrenal bleeding in a patient who developed shock before death could be directly related to envenoming or a secondary manifestation. Nevertheless, there are a few cases of bleeding tendency reported in the past.[19] A significant number of patients had myalgia but the lack of facilities prevented us testing for myoglobin in plasma and urine. A former study of five common krait bites at Anuradhapura had demonstrated myoglobinaemia in one patient who had myalgia.[7] One of the patients envenomed by the Malayan krait (*Bungarus candidus*) had generalised muscle pain and tenderness.[20] These suggest that presynaptic phospholipase A2 could cause rhabdomyolysis. However, none of the present patients had renal consequences of rhabdomyolysis. The question is: Does a similar process affect the smooth muscles in the stomach and gut to produce abdominal pain and minute bleeding?

Severe respiratory paralysis was seen in the half the number of patients and they would have died without assisted ventilation. Similar results have been reported in a previous study in which two patients out of five needed assisted ventilation, and it gives us an understanding about survival chance in natural evolution of envenoming in common krait bite.[7] Soon after establishing the intensive care unit at Anuradhapura, a study was done in a series of 30 patients who were managed with assisted ventilation in the year 1990 and the duration of ventilation ranged from eight hours to 10 days (median 33 hours), which is comparable with the duration of the present series.[11] Rapidity of the development of respiratory paralysis in the present study showed a wide range from 30 minutes to 50 hours and another study has shown time range from seven to 12 hours.[15]

On comparison with cobra bite, which can kill a man in 30 minutes, the progression of envenoming may be slow in an occasional case of krait bite.[1 7]

The phenomenon of hypokalaemia needs explanation. A previous study has observed hypokalaemia in three patients and it was thought to be due to respiratory alkalosis as a result of hyperventilation.[11] Hypokalaemia was considered as an indicator to adjust the rate and volume of respiration in assisted ventilation.[11] However, the incidence of hypokalaemia was very high in the present study and it was associated with metabolic acidosis even though there were normal blood gases. The most likely explanation is the internal shift of potassium into cells due to β-adrenergic stimulation as a result of autonomic dysfunction.[21] However, external losses and internal shift of K^+ and H^+ due to hormones such as insulin, renin, and aldosterone should be excluded.

Delayed neuropathy is different from acute neuromuscular paralysis and needs structural damage to nerve fibre, nerve ending, or demyelination. A case report has highlighted symmetrical distal motor neuropathy after Ceylon krait bite in 1988.[22] The neurotoxins probably cause ultrastructural damage to motor nerve endings.[23] Excitation of both parasympathetic and sympathetic autonomic systems explains most of the clinical manifestations like increased secretions, dilated pupils, tachycardia, and hypertension. A patient who had a Malayan krait bite had fixed dilated pupil, sweating, tachycardia, and hypertension due to parasympathetic abnormalities.[20]

Alteration of the sensorium and progression to a deeply comatose state is not simply explained by cerebral hypoxaemia and the locked-in position due to severe neuromuscular paralysis. These patients were well oxygenated and the brainstem function tests were negative. Furthermore, associated anterograde memory loss is strongly suggestive of widespread depression of cerebral functions. Documentation of these observations are few in the literature. One patient with Ceylon krait bite remained deeply unconscious until death.[12]

The polyvalent antivenom, which is manufactured in India, has doubtful efficacy in reversing established neuromuscular and respiratory paralysis.[7 8 17] Theakston *et al* clearly show the clearance of venom antigenaemia with intravenous polyvalent antivenom.[7] It had no effect on bound antigen in neuromuscular junctions, which occurs quite rapidly with envenoming, but it neutralises the unbound venom in the blood. This concept is supported by the absence of correlation between recovery of respiration and dose of administered antisera observed in the study. Development of highly penetrable monospecific antivenom against the local species to counter bound venom might reverse prolonged neuromuscular paralysis. Finding a suitable drug to displace venom tagged to the neuromuscular junction has been considered by many workers. Anticholinesterase had been tested and no benefit was found in reversing paralysis in two common krait bites and it was further confirmed by neurophysiological tests in six patients in a recent study.[7 15] However, anticholinesterase had produced dramatic improvement in one patient after Malayan krait bite.[20]

Neurotoxins of the Asian cobra are predominantly postsynaptic in action and their effect can be reversed by the administration of anticholinesterase.[24] Therefore, development of a new drug with presynaptic action might be useful in common krait bites, rather than further testing anticholinesterase.

The overall picture so far discussed is beyond the action of presynaptic neuromuscular block by β-bungarotoxin, phospholipase A2. Studies report the existence of 16 isotoxins in the β-bungarotoxin family.[25 26] Anticoagulant and neurotoxic activities were found in the protein isolated from common krait venom which was subjected to sequence and crystal structure determination.[27] The enzyme phospholipase A2 is shared by many snakes and it has a wide array of effects including haemolysis, vasodilatation, rhabdomyolysis, and release of endogeneous autocoids in addition to neuromuscular blocking.[28] Further studies into venom toxicology would facilitate the understanding of the overall picture of krait bite.

This study identified factors such as poor resuscitation, delay in hospital admission, and complications contributing to deaths in common krait bite in Sri Lanka. Improvement of intensive care facilities in hospitals, awareness of the clinical course, and anticipation and management of complications can overcome the morbidity. Educating high risk populations about the biting pattern of the common krait and on preventive measures is likely to reduce the incidence of snake bite.

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